without understanding fully that there is some risk of inducing a lasting anosmia. Inasmuch as the effect of the drug in almost any concentration down to and including one-tenth of one per cent is likely to be influenced by the anatomic configuration of the nasal passages, as well as by other factors difficult to control, it seems highly improbable to me now that the application of zinc sulfate solution can be controlled sufficiently well to insure both complete freedom from the risk of permanent anosmia and full protection. We already know that a one per cent solution of zinc sulfate does induce a lasting anosmia in some persons,[9] possibly in one or two per cent of those treated, and that adults are more especially prone to such a complication, probably largely for local anatomic reasons.[10] At the same time I am sure that this risk can be reduced considerably by a somewhat more cautious procedure in applying the solution, the principle features of which should be to begin with a much lower concentration of the drug, and to restrict its action by some appropriate means.[11] It is known now that the most effective way to apply a solution to the olfactory area is by instilling it slowly along the nasal sulcus, while the head is being held in the fully inverted position.[12] The same method, therefore, should also facilitate the removal or dilution of the drug by the subsequent introduction of saline solution.

### PRACTICAL CONSIDERATIONS

From the practical standpoint, it is important to bear in mind that, while we wish to avoid permanent impairment of the sense of smell, the induction of a temporary anosmia is probably necessary to convey protection. By the same line of reasoning, a return of the sense of smell probably means that susceptibility to infection has also returned. To attain just this degree of impairment, but no more, it would seem desirable to proceed from a very low concentration to one which will just induce anosmia (minimal anosmic dose). I should, therefore, suggest starting such a series of treatments with a concentration of the drug as low as, or lower than one-tenth of one per cent, and increasing it in subsequent treatments, administered at intervals of two or three days, by not more than 0.1 per cent, until an anosmia, as determined by a dependable method, has been induced. Until we have a more extensive background of experience, drawn from observations on man himself, it will not be possible to say how much this more cautious approach may be exceeded. Whatever steps can be taken to eliminate the risk of a lasting anosmia, it will not be easy to determine the prophylactic effectiveness of such a measure, and it may require many years before its actual practical value in man can be determined. Obviously, studies such as these should not end with zinc sulfate and are, in fact, being continued with the hope that something may eventually be found which will prove less irritating and less hazardous to the sense of smell.

### SPECIFIC THERAPY

Regarding specific therapy during the acute stage of the disease, nothing constructive has as yet emerged from the experimental laboratory. However, studies are now in progress to determine whether the experimental disease can be made to yield to some specific chemical agent, either by virtue of a direct action on the virus or by virtue of a helpful physiologic action on the infected host. The results of such investigations cannot yet be predicted.

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## POLIOMYELITIS*

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THE importance of the poliomyelitis problem and its intensive study justifies an attempt to correlate the acquired data, particularly concerning (1) the carrier; (2) the criteria for early diagnosis, with recognition of the nonparalytic case; and (3) the evaluation of spinal-fluid findings.

### ETIOLOGY

Most epidemiologists agree that control could be effected were rapid identification and isolation of the carrier possible. Unfortunately, this depends on some as yet undiscovered chemical, serological, or other method of virus identification more applicable than the present slow, uncertain, or cumbersome biologic tests. Rosenow's[1] streptococcus, morphologically and cataphoretically identified, seems to fulfill Koch's postulates; and Eberson and Mossman's[2] culture of a microscopically visible, biologically identified, organism likewise seems convincing, but the probable existence of extrinsic factors remains.

The Swedish Commission's[3] report is replete with significant data. These scientists showed, first,

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that the virus is relatively large and frequently will not pass the ordinary Berkfeld-W candle filter in amounts sufficient to effect successful monkey inoculation with a reasonable volume of material; and, second, that the Macacus cynomolgus monkey is much more susceptible than the commonly used Macacus rhesus. They repeatedly demonstrated virulent virus in small-volume washings from intestinal mucosae of convalescents, in some instances many months after clinical recovery. Because their work was done early in the experimental investigation of the disease, and they did not realize the importance of demonstrating the pathologic lesion known as neuronophagia in all instances, their work has been somewhat discredited. In the light of ensuing and more recent investigation, it is beginning once more to assume its proper importance. Very recently Trask, Paul, and Vignec[4] recovered virus from the human stool, and Osgood and Lucas report carriers as long as two years after clinical recovery. This possibly explains the frequency of relapse[5]—by autoreinfection during periods of diminished acquired immunity—and assists in clarifying the morbidity among certain groups of hospital personnel reported in the Los Angeles epidemic[5] of 1934.

That convalescents constitute a prolific carrier source seems a logical hypothesis. The nonparalytic (abortive) case is, likewise, dangerous, for its rapid recovery is evidence of adequate immunogenic response. This was proved serologically by Howitt,[6] when she demonstrated higher antiviral properties in serum from spontaneous convalescents; and in a striking fashion clinically by Clauss Jensen,[7] when he used serum from abortive cases and recent convalescents with remarkable therapeutic success.

The atrium, by which virus reaches the central nervous system, is by no means a closed question. The olfactory pathway is considered the common avenue, and Schultz and Gebhardt[8] have proved that it is at least one portal. They sectioned the olfactory nerves of monkeys; then failed to infect them with intranasal implants of virus, subsequently proved potent by intracerebral inoculation. However, unsuccessful nasal implantation loses much significance when it is remembered that in nature poliomyelitis is limited to the human host, and infection of animals by contact methods is always difficult. In fact, Harmon, Shaughnessy and Gordon,[9] and many others have failed to produce a clinical picture of poliomyelitis simulating that in the human, with recovery of the virus, in any animal except monkey, and in the latter nasal infection is usually very difficult. The suggested existence of a virus neutralizing substance in the nasal mucosa[10,11] seems improbable in view of the recovery of unneutralized virus in nasal washings from monkeys infected by other routes. It seems logical to deduce that the nasal barrier is mechanical and that these membranes can constitute an *efferent* avenue for egress of virus, as well as a probable *afferent* portal for its entry.

That the gastro-intestinal tract may be a common atrium of infection in man is the contention of many observers. The cumulative evidence of involvement of all reticulo-endothelial tissues in the early, systemic phase of the disease, and the uniform and marked changes in the splanchnic lymphatics seem to incriminate the gastro-intestinal tract. Much of the brilliant work of the Swedish investigators[3] was by intraperitoneal and sciatic nerve injection of small amounts of filtrate from rectal washings of patients, and the virus thus obtained was identical with that found in the nasal mucosae. Toomey,[12] after extensive investigation, including cord transection,[13] quite logically considers the perineural lymphatics, or nerve sheaths of the sympathetics, as a probable atrium to the central nervous system. Intraperitoneal injection suggests also an indirect approach through the general lymphatic channels. Brodie and Elvidge[14] failed to substantiate Toomey's results; but Landon and Smith[15] reporting the largest recorded series of human autopsies (ninety-six), concur in the opinion that the gastro-intestinal tract is a probable atrium of infection in man.

Clinically, we have seen many cases with early gastro-intestinal symptoms; secondary involvement of the lower extremities; and a complete absence of respiratory manifestations. The inference is obvious.

Another potential method of infection is through breaks in the skin. In our hospital, Kessel has repeatedly produced experimental poliomyelitis in monkeys by intradermal injections of minute quantities of one strain of poliomyelitic virus.

Considering the above factors in virus dissemination, it would seem advisable ultimately to require reasonable proof of noninfectiousness as a criterion for release from quarantine. The absurdity of releasing diphtheria or typhoid patients after a fixed quarantine period, and regardless of bacteriologic findings, is obvious, and the analogy in the case of poliomyelitis is evident.

## PATHOLOGY

A brief review of the demonstrable pathology of poliomyelitis may explain certain apparent clinical discrepancies. Landon and Smith[15] call attention to the hyperemia and perivascular round-cell infiltration; capillary thrombi; infarction and necrosis in liver, spleen, Peyer's patches, tonsils, thymus, heart, and endocrine glands. The thymus is more consistently enlarged as a pathologic entity in this disease than in any other known condition, except status thymicolymphaticus. Peyer's patches resemble the condition seen during the first week of typhoid fever. Our Swedish colleagues,[3] and Burrows,[16] have called attention to the generalized lymphatic hyperplasia. While the poliomyelitic virus has *known* neurotropic propensities, *it is also evident that it produces a systemic infection.* This explains the early clinical picture of generalized toxemia.

Meningeal exudate was shown by Landon and Smith[15] to be present only in 70 per cent of their ninety-six autopsied cases. Our own and other observations are corroborative. The pathology of poliomyelitis is not primarily involvement of the meninges, or of the choroid; hence the frequent paucity of cells in the spinal fluid is no enigma. The

earliest evidence of nervous system involvement is frequently periradicular infiltration and piarachnoid engorgement, hence it is difficult to understand why the profession at large is so reluctant to consider anything short of demonstrable paralysis as constituting clinical poliomyelitis; or why, in the face of a clean-cut clinical picture, such undue importance is attached solely to spinal-fluid findings in all cases.

Microscopic study of the central nervous system in poliomyelitis discloses facts quite at variance with the general anatomical-clinical concept. In autopsied cases, instead of lesions being localized solely to the anterior horn cells which innervate the clinically affected muscles, these areas of inflammation extend diffusely throughout the midbrain, basal ganglia, pons, medulla and cord, varying only in degree. To what extent this is true in milder cases is conjectural, but the parallelism should hold proportionately.

Two main features are noted in the motor cells: (1) early death without degeneration, but with demonstrable microscopic changes; and (2) late neuronophagia, the replacement of the degenerated cell by microglia, the essential scavengers of the central nervous system. There is also a diffuse interstitial infiltration by these Hortega[17] cells (microglia) throughout the cord, including the lateral and posterior horns and the fasciculi; in very severe cases actual necrosis occurs. Perivascular round-cell infiltration is also present, while edema of the cord is more marked than in the brain, but degeneration appears to be independently related to the direct action of the virus on individual cells.

Considering the clinical severity of cases coming to autopsy, the gross pathology seems remarkably slight.

## EARLY DIAGNOSIS

Early diagnosis, and particularly the recognition of nonparalytic (abortive) cases, is important if early therapy, isolation, and carrier detection are to be effected. The early toxemia simulates that of other acute infections; hence, justifiable diagnostic errors are common. One may not ignore, or fail to elicit, the finer points in differential diagnosis.

During the stage of invasion, the fever, headache, generalized muscle pains and tenderness, hyperesthesia and varying degrees of gastro-intestinal disturbance are similar to many infectious diseases. While constipation is usually mentioned, frequently we have observed an early and marked diarrhea. Even some degree of meningism is not uncommon, and the characteristic headache of poliomyelitis is not pathognomonic—though very significant in little children. Upper respiratory infections involving any of the perinasal sinuses, or the peripheral nerves, may cause similar pains in the frontal region; and meningism, when present, may produce cellular changes in the spinal fluid quite as marked as those of poliomyelitis. However, poliomyelitis is notoriously likely to produce *clinical* findings referable to isolated groups of nerve cells despite the diffuse pathology noted at *autopsy. This tendency to produce localized clinical findings is the key to early diagnosis.* Poliomyelitis very early shows isolated muscle tenderness, isolated muscle weakness, asymmetry of reflexes, or rapidly changing reflexes. Early, for some unexplained reason, it is common to find an absence of the superficial reflexes, those of the deep tendons being often exaggerated during the irritative, invasion stage. Later, asymmetry or absence of the deep reflexes occurs, if the case progresses. The spine sign, Brudzinski, and Kernig may become present.

The spinal fluid may show no changes at any time, particularly in nonparalytic (abortive) cases, although the abnormally high antiviral titer of the serum of these individuals, as shown by Howitt[6] and Jensen,[7] furnishes the needed final proof of their infection with poliomyelitic virus.

The late findings in poliomyelitis are so well known that we will mention only the more remote sequelae.[5] Emotionalism, lack of acuteness of attention and decreased power of concentration are the most common encephalitic phenomena. Parkinsonian syndrome occurs rarely. Localized hirsutism, changes in pigmentation, localized hyperhidrosis, etc., have also been mentioned,[5] and clinically confirm Toomey's[12] hypothesis of sympathetic nervous system invasion. Involvement of the endocrine system is also definitely indicated, and in some cases becomes severe.

In a number of cases occurring since May, 1934, and of several years' duration, we have observed recurrent attacks of purpura at relatively frequent intervals. We have found no mention of this phenomenon in the literature. Usually accompanying these purpuric episodes are headache, one or two degrees of fever, return of muscle tenderness and pain, and a temporarily diminished muscle strength as demonstrated by checking affected groups. Macules vary from a pinhead to a split-pea size.

## SPINAL FLUID IN POLIOMYELITIS

The frequent absence of an increased spinal fluid cell count has been discussed.

Following the 1930 epidemic in Los Angeles,[18] we reported an absence of cellular changes in the spinal fluid in 12.6 per cent of clinically proved cases of poliomyelitis. Skeptics cast doubt on this finding.

Following the 1934 epidemic in Los Angeles, we reported[4] similar findings in approximately 33 per cent of 1,800 cases. In the same year Clauss Jensen[7] reported no spinal fluid change in 36.1 per cent of 3,340 cases seen in Denmark. Landon and Smith,[15] Brahdy and Lenarsky,[19] Brodie and Wortis,[20] and others, also admit similar findings occasionally. With these corroborative reports, and our additional experiences in a larger epidemic, we feel fully vindicated concerning the original contention that, at the present time, one must be prepared to diagnose poliomyelitis from the history and physical findings, *in the absence of cellular changes in the spinal fluid.* We cannot concur with Herrick's[21] statement to the effect that during an an epidemic the final diagnosis depends entirely on the spinal fluid. The above references to negative spinal fluids in clinically proved cases, to autopsy findings, and to the recovery of virus in respiratory

or intestinal washings from nonparalytic (abortive) cases seem adequate proof to the contrary. The pathology of poliomyelitis usually does not include primary meningeal or choroid involvement, and any increased spinal fluid cell count is only an index of subsequent meningeal invasion; absent in at least 30 per cent of autopsied cases.

### DIAGNOSIS

Early direct diagnosis of the nonparalytic case has been considered above, and the paresis or paralysis occurring later needs no discussion. Differential diagnosis at times must include practically all acute infections, particularly those of the respiratory or gastro-intestinal tracts showing initial toxemia. The list is too long to reiterate. In 1935,[5] we mentioned fifty-seven different conditions misdiagnosed as poliomyelitis, many of them justifiably. In addition, the list now includes one case each of tetany, trichiniasis, encephalomyelitis from neoarsphenamin, acute lead poisoning, and septic cerebral embolus in a child suffering from acute bronchopneumonia. Practically all conditions of the central nervous system may require differential diagnosis.

### TREATMENT

Prophylactically, we maintain a conservative attitude. The vaccines of Brodie[22] and Kolmer[23] are similar to those of Levaditi and Landsteiner,[24] which were shown to be inert in the one case and dangerous in the other. One of our fatal cases was a man "immunized" less than one month previously. Another less severe case was a nurse similarly "immunized" six months before. At the present time we believe that the hazards of vaccination are minimal but actual, and that its benefits are still debatable. We have in the past used nonspecific autohemotherapy on the hypothesis that immunity depends largely on reticulo-endothelial response, and that the autolysis of whole blood may constitute a normal biologic stimulant. We have no convincing data, but believe that Waltner's[25] controlled series is significant. We are not overenthusiastic about passive immunization, although we are inclined to agree with the statement Kolmer made in 1938 at the annual convention of the American College of Physicians, that injection of high titer convalescent serum is probably the best medium we possess at present. Improperly controlled, all immunization programs may be actually dangerous, and the truth lies in the future.

Nasal sprays are still experimental and seem to offer little more than doubtful, transient protection: they endanger the olfactory nerve, and may destroy it at times.

Serum therapy is still a disputed question. Again we cite the very general agreement of laboratory workers concerning the *in vitro* and *in vivo* antiviral properties of convalescent serum. Our statistics[18] on the 1930 epidemic seem most significant, the serum-treated hospital cases showing 3.2 per cent mortality as against 7.6 per cent for the entire county during the same epidemic. However, other observers fail to show such a difference, and our results with serum were much less striking in the 1934 epidemic. We may have been dealing with different strains of virus, such being known to exist, and probably a great deal of the serum used possessed no protective properties.[26] Jensen's series[7] is a strong argument in favor of serum therapy. Following his lead, at the present time all serum prepared at the serum depot in the Children's Hospital in Los Angeles is pedigreed, that which will not prevent infection in monkeys being discarded.

The method of administration is also debatable. It would seem logical to replace serum in its own element, intravenously; particularly in view of the recognized systemic phase of the disease and the rapidity of distribution offered by this route. Intramuscular injection allows of slower absorption over a longer interval. Intrathecal administration by lumbar or cisternal routes is still more debatable. Theoretically, little good should be expected; yet in 1930, after instituting cisternal administration, we lost few of the relatively large number of cases showing clinical involvement high in the central nervous system: prior to adopting the procedure most of these patients were dying. The immediate amelioration of symptoms following intrathecal puncture may be hydrostatic, relieving perineural edema by shifting the osmotic balance between the capillary circulation and the spinal fluid.

Immunotransfusion[27] offers dual possibilities because it combines antiviral specificity with the known beneficial effects of hemotherapy. Where proved donors are available, we prefer it. Results are occasionally very spectacular.

A series of cases treated with intravenous hypertonic dextrose—10 per cent in normal saline[5]—showed decided merit for this therapy. Autopsies on dextrose-treated patients have shown occasional capillary thrombi, but the value of this therapeutic agent in mobilizing reticulo-endothelial defenses, promoting renal elimination, and stimulating hepatic function—detoxication, glycogenesis, antiketogenic glycogenolysis—is too well established to be discredited.

Retan [28] successfully used hypotonic solutions intravenously, with frequent withdrawal of excess spinal fluid. We have not tried his method in this disease. The question of hyper- or hypotonic solutions is only one of osmosis, with attempted washing of virus in different directions and utilization of different escapes.

Based upon the work of Meltzer,[29] 0.5 to 1.0 cubic centimeter of 1:1000 solution of epinephrin hydrochlorid was given intraspinally by Hoyne[30] in 1916 with apparently good results. We have had no experience with the procedure. The ephedrin treatment later advocated by Royle,[31] on the basis of reducing destructive edema by facilitating capillary circulation, has not been tried in a large controlled series. Courville, in Los Angeles, suggested its use independently in 1934, but the exigencies of the epidemic prevented running a controlled series. It was tried in a few cases without obvious benefit.

### SUMMARY AND CONCLUSIONS

Evidence is herewith presented to show that:

1. Carriers of poliomyelitic virus definitely exist, and are important factors in the dissemination and control of poliomyelitis.

2. Sera from *recent, rapidly convalescent and nonparalytic patients* show high antiviral titers: these individuals are most prolific sources of carriers.

3. Human vectors harbor the virus in their intestinal and respiratory tracts.

4. Identification of virus, and hence detection of carriers, is by means of time-consuming, expensive and difficult biological tests, and is uncertain at present.

5. Proof of noninfectiousness ultimately must be the criterion for release from quarantine.

6. Studies in pathology show that (*a*) poliomyelitis is a systemic disease primarily, and a central nervous system disease secondarily; (*b*) the systemic phase usually precedes or accompanies the central nervous system phase, which may be absent altogether; (*c*) the nervous lesions are diffuse below the midbrain, in spite of apparent clinical localizations; (*d*) the degree of pathologic change in the central nervous system does not parallel the clinical picture; (*e*) the meningeal involvement is only commensurate with the degree of systemic infection, and may be absent entirely; (*f*) cellular changes in the spinal fluid are proportional to the degree of meningitis present; and (*g*) the spinal fluid cell count may remain entirely normal and unchanged.

7. Diagnosis in many instances does not depend on spinal fluid corroboration.

8. Recognition of the nonparalytic case is possible and most desirable, as this individual is a latent source of potential infection.

9. Present prophylactic vaccines offer encouragement, but no convincing proof of immunity, and Kolmer admits the morbidity is greater in the vaccinated group than in the unvaccinated population.

10. Immunotransfusion is recommended early in severe cases.

11. Convalescent serum by all routes has proved beneficial in our experience.

12. Hypertonic dextrose in normal saline intravenously has proved advantageous in the systemic phase of the disease, and Retan believes the hypotonic solution saves lives in the paralytic group when given by his method.

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## POLIOMYELITIS: ITS TREATMENT*

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THE treatment of poliomyelitis is an ungrateful subject for discussion, for it has taken us many years to appreciate the importance of a few measures, proved useful, essential to the proper care of the patient. These measures are so few and so simple that a discussion of treatment should best reiterate and reëmphasize them. A vast amount of scientific study of this disease has been productive of a pathetic paucity of information regarding specific curative efforts which can serve as basis only for speculation as to the future of treatment.

We may properly divide our discussion into three phases: (1) the essentials of management of the typical attack apart from paralytic manifestations; (2) the treatment of the various forms of muscular

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